UNITED STATES DISTRICT COURT
NORTHERN DISTRICT OF INDIANA
SOUTH BEND DIVISION

C.W., et al.	)	
	)	
Plaintiffs,	)	
v.	)	
	)	NO. 3:10 CV 87 PPS
TEXTRON, INC.,	)	
	)	
Defendant.	)	
	)	

OPINION AND ORDER

Before me are cross motions to exclude a number of experts that each party has retained in this toxic tort case involving groundwater contamination. In addition to the parties' voluminous submissions, I conducted a two-day hearing on the admissibility of the experts' testimony. Following the hearing, I have determined that the testimony of the Plaintiffs' experts regarding causation of their medical problems is not sufficiently reliable and must be excluded. Because this opinion may be case dispositive, I also dismiss the remaining pending motions to exclude expert testimony as moot.

BACKGROUND

Plaintiffs, C.W. and E.W., are the minor children of Adele and Jason Wood. (For simplicity's sake, I will refer to the Plaintiffs as "the Woods") They allege that the water they used in their Rochester, Indiana home was contaminated with a chemical called vinyl chloride ("VC"), which seeped into the grounwater from a facility owned by Textron.

C.W. was adopted and brought to the Wood home on May 11, 2007, when he was 11 weeks old. E.W. was adopted and brought to the home on April 25, 2008, when she was 11 days old. The Woods have alleged that the children were exposed to the vinyl chloride, a known

human carcinogen, by ingestion of the drinking water, from dermal contact, and through vapor inhalation when they were bathed. The Woods brought both of the children to their Rochester home before they were aware of the contamination, which they were notified of on November 5, 2008. The children were removed from the home that day, and have not returned.

Textron began monitoring its Torx facility in the 1980s, when onsite monitoring wells began to show increased levels of organic compounds. From 1988 to 2008, Textron submitted monitoring reports to the Indiana Department of Environmental Management, or IDEM, without incident. In November 2008, groundwater contamination was discovered in offsite wells. Specifically, Textron consultants performed testing at the Woods home on November 3, 2008, and found that their water contained 5.92 micrograms of vinyl chloride per liter of water. A test three days later showed a concentration of 5.00 micrograms per liter of water. On November 18, 2008, IDEM collected a sample that showed vinyl chloride at a level of 8.4 parts per billion. Finally, Textron's consultants conducted additional testing at the home in September 2009, which resulted in vinyl chloride concentrations of 9 parts per billion and 8.6 parts per billion.

To understand the dosage, one needs to understand the math: one microgram per liter is also known as one part per billion, or "1 ug/l." This means, for example, that the reading taken from the Wood home on November 3, 2008 represented 5.92 parts per billion or 5.92 ug/l.

The Woods claim that vinyl chloride has caused a number of illnesses in their children and substantially increased their risk of adverse health effects throughout their lives. The Woods allege that while the children lived in the Rochester home, they suffered from gastrointestinal issues, including spitting up, projectile vomiting, and bloody stools; that E.W. had skin problems in the form of an itchy rash; and that both children have suffered neurological problems. They

also contend that the children are now at an increased risk for cancer. In support of these allegations, they have proffered the expert testimony of a number of experts as to causation, and specifically have offered the testimony of three doctors to support their claims: Dr. Jill Ryer-Powder, Dr. Vera Byers, and Dr. Jeffrey Dahlgren.

Dr. Ryer-Powder is a toxicologist who proposes to testify that exposures to VC were at levels sufficient to cause harm to E.W. and C.W. and that these harms include a future increased risk of cancer. Textron objects to this testimony on several bases: first, they contend that Dr. Ryer-Powder's reliance on exceedance of regulatory standards is flawed [DE 142 at 9]. They also argue that her dosage calculations overstate the Woods' actual exposure to vinyl chloride [*Id.*]. Third, and the principal objection that I will address, is Textron's contention that the studies that Dr. Ryer-Powder relies upon to support her conclusion that the Woods' VC exposure is sufficient to cause an increased risk of cancer involve levels of exposure far greater than are present in this case, and that Dr. Ryer-Powder fails to explain the basis for extrapolating these results to apply them to this case [*Id.*].

The Woods have also proffered the testimony of Dr. Vera Byers, an immunologist. Dr. Byers proposes to testify that due to their exposure to vinyl chloride, the children have an increased risk of cancer, immunological deficiencies, and gastrointestinal issues. Textron objects to her testimony for reasons similar to their objections to Dr. Ryer-Powder: her opinion regarding general causation is not supported by scientific research or a reasoned basis for extrapolating high dose worker studies to this case; her differential diagnosis is flawed; and to the extent that she relies upon other challenged experts whose testimony is inadmissible, her opinions should also be barred [DE 155 at 1-2].

Textron also challenges the testimony of Dr. Jeffrey Dahlgren. Dahlgren proposes to testify that as a result of their exposure to vinyl chloride, the Wood children experienced gastrointestinal, respiratory, neurological, and immunological problems, and that they are highly likely to be diagnosed with cancer in the future [DE 146-1]. Among other objections, Textron argues that Dr. Dahlgren's methodology relating to causation is flawed because it improperly relies upon regulatory standards; his opinions are unsupported by scientific research; and because his differential diagnosis is flawed [DE 157 at 2-3]. Textron also argues that to the extent that Dahlgren relies upon the opinions of other challenged experts, his opinions are subject to exclusion on the same grounds [DE 157 at 3].

I will start with the law that governs these motions and then turn to the objections to each of the proposed experts.

DISCUSSION

The admissibility of expert testimony is governed by Federal Rule of Evidence 702 which was substantially amended after *Daubert v. Merrell Dow Pharm.*, 509 U.S. 579 (1993). Federal Rule of Evidence 702 states:

> A witness who is qualified as an expert by knowledge, skill, experience, training, or education may testify in the form of an opinion or otherwise if: (a) the expert's scientific, technical, or other specialized knowledge will help the trier of fact to understand the evidence or to determine a fact in issue; (b) the testimony is based on sufficient facts or data; (c) the testimony is the product of reliable principles and methods; and (d) the expert has reliably applied the principles and methods to the facts of the case.

The *Daubert* framework requires a district court to determine whether the expert's testimony is both reliable and relevant. In particular, the district court must determine whether

(1) the proposed witness would testify to valid scientific, technical or other specialized knowledge; and (2) his testimony will assist the trier of fact. *Ammons v. Aramark Unif. Servs., Inc.*, 368 F.3d 809, 816 (7th Cir. 2004). The "reliability" factor includes a determination of "whether the expert is qualified in the relevant field and whether the methodology underlying the expert's conclusions is reliable." *Id.* (citation and internal quotations omitted). Anyone with relevant expertise enabling him to offer responsible opinion testimony helpful to the jury may qualify as an expert witness. *See* Fed. R. Evid. 702; *United States v. Navarro*, 90 F.3d 1245, 1261 (7th Cir. 1996).

Rule 702 and *Daubert* make me the "gatekeeper" with respect to the screening of expert testimony. *Kumho Tire Co., Ltd. v. Carmichael*, 526 U.S. 137, 147-48 (1999) (citing *Daubert*, 509 U.S. at 589); but as the Seventh Circuit recently explained, ". . . the key to the gate is not the ultimate correctness of the expert's conclusions. Instead, it is the soundness and care with which the expert arrived at her opinion: the inquiry must 'focus . . . solely on principles and methodology, not on the conclusions they generate.'" *Schultz v. Akzo Nobel Paints, LLC*, 721 F.3d 426, 431 (7th Cir. 2013) (citing *Daubert*, 509 U.S. at 595).

Because experts come in all shapes and sizes, the *Daubert* framework is necessarily a flexible one that must be adapted to the particular circumstances of the case and the type of testimony being proffered. *Mihailovich v. Laatsch*, 359 F.3d 892, 919 (7th Cir. 2004). As the Supreme Court elaborated, the objective "is to ensure the reliability and relevancy of expert testimony. It is to make certain that an expert, whether basing testimony upon professional studies or personal experience, employs in the courtroom the same level of intellectual rigor that characterizes the practice of an expert in the relevant field." *Kumho*, 526 U.S. at 152. District

judges are afforded substantial discretion in making these determinations. *Roback v. V.I.P. Transp., Inc.* 90 F.3d 1207, 1215 (7th Cir. 1996).

Generally speaking, courts are to consider four non-exclusive factors in assessing the reliability of an expert witness: "(1) whether the scientific theory can be and has been tested; (2) whether the theory has been subjected to peer review and publication; (3) the theory's known or potential rate of error when applied; and (4) whether the theory has been 'generally accepted' in the scientific community." *Fuesting v. Zimmer, Inc.*, 421 F.3d 528, 534 (7th Cir. 2005) (citing *Daubert*, 509 U.S. at 593-94), *vacated on other grounds*, 448 F.3d 936 (7th Cir. 2006). But as alluded to above, because not all expert testimony can be neatly examined under the factors set forth in *Daubert*, the factors are "neither definitive nor exhaustive, but rather flexible to account for the various types of potentially appropriate expert testimony." *Deputy v. Lehman Bros., Inc.*, 345 F.3d 494, 505 (7th Cir. 2003) (citing *Kumho*, 526 U.S. at 141). Thus, the *Daubert* factors may not apply in every instance.

In toxic tort cases, there is a two-step process in examining the admissibility of causation evidence. First, the district judge must determine whether there is general causation, and if there is, then the judge must determine whether there is admissible specific causation evidence. *Knight v. Kirby Inland Marine, Inc.*, 482 F.3d 347, 351 (5th Cir. 2007). General causation "establishes whether the substance or chemical at issue is capable of causing a particular injury or condition in the general population;" by contrast, specific causation "establishes whether the substance or chemical in fact caused the plaintiff's medical condition." *Baker v. Chevron USA, Inc.*, 680 F. Supp.2d 865, 874 (S.D. Ohio 2010); *see also Aurand v. Norfolk Southern Railway Co.*, 802 F.Supp.2d 950, 953 (N.D. Ind. 2011). "Scientific knowledge of the harmful level of exposure to

a chemical, plus knowledge that the plaintiff was exposed to such quantities, are minimal facts necessary to sustain the plaintiffs' burden in a toxic tort case." *Allen v. Pa. Eng'g Corp.*, 102 F.3d 194, 199 (5th Cir. 1996). In assessing the reliability of these experts' methodology and testimony under *Daubert*, "I must carefully review the specific sources and scientific results relied on by [them] to determine whether they appear to be valid and reasonably applicable to support [their] causation opinion[s]." *Aurand*, 802 F. Supp. 2d at 954.

With these principles in mind, I turn to the opinions of each of the Woods' three causation experts and the objections to their testimony raised by Textron.

I. Dr. Ryer-Powder

Dr. Ryer-Powder prepared two reports in this case: the first, dated August 17, 2011, laid out her opinions that:

> 4. Exposures to [C.W. and E.W.] were at levels sufficient to cause harm during the exposures; and
>
> 5. Exposures to [C.W. and E.W.] were at levels sufficient to present an unacceptable risk of cancer in the future.

[DE 147-1 at 6]. The initial report contained a list of references in Appendix C [DE 147-1 at 35-37].

Following the filing of Textron's motion to exclude her report and testimony, Dr. Ryer-Powder issued a "rebuttal" report[1], dated November 18, 2012 [DE 168-1]. The report responded

[1]Dr. Ryer-Powder, Dr. Byers, Dr. Dahlgren, and two of the Plaintiffs' other experts who are not at issue here each provided supplemental reports that Plaintiffs used in responding to Textron's *Daubert* motions. Textron has moved to strike these reports as untimely and improper [DE 212]. I have considered these supplemental reports in this opinion, and because I am granting Textron's motions to exclude these experts, I will **DENY** this motion [DE 212] as moot.

to the motion to exclude and addressed each of Textron's objections to her testimony. Specifically, it argues that government regulations are developed by consulting "primary literature upon which they are based," and that "[d]ocumentation includes the use of toxicity studies published in the primary, peer-reviewed literature," and that such literature demonstrates "a marked increase in the incidence of liver cancers in newborn animals compared to adult animals exposed to vinyl chloride." [DE 168-1 at 2]. As to Textron's argument that her dosage calculation is flawed, she opined that her dosage calculations were proper based on IDEM water readings [DE 168-1 at 3]. Finally, she opined that scientific literature demonstrated that "doses of vinyl chloride administered to animals that caused liver tumors are within 10 times those of the Wood children" and that primary studies show that newborns have an increased susceptibility to the carcinogenic potential of vinyl chloride [DE 168-1 at 2].

I will begin with Textron's first objection to Dr. Ryer-Powder, her reliance on regulatory standards. Textron is correct that this alone is an improper basis for an expert opinion, for mere exposure to toxins in excess of regulatory levels is insufficient to establish causation. *Cunningham v. Masterwear Corp.*, 569 F.3d 673, 674-75 (7th Cir. 2009); *Baker*, 680 F. Supp. 2d at 880 (". . . to the extent that Dr. Dahlgren relies on the fact that Plaintiffs' illnesses were caused because they were exposed to benzene in excess of regulatory levels, his opinions are not admissible"). The rationale for this is understandable: "regulatory agencies are charged with protecting public health and thus reasonably employ a lower threshold of proof in promulgating their regulations than is used in tort cases." *Baker*, 680 F. Supp. 2d at 880 (citing *Allen*, 102 F.3d at 198.

The Woods, without citing any case law that supports their argument that exceedance of regulatory standards is appropriate in a causation analysis, resort to a "what's sauce for the goose is sauce for the gander" argument, which is essentially that Textron's experts have, to some extent, conceded that exceedance of regulatory standards plays into an evaluation of the harm that vinyl chloride could cause [DE 168 at 4-6]. It may be the case that regulatory exceedances, in combination with other evidence, can be relied upon in forming causation opinions. But if the expert's causation opinion is based *solely* on exceedance of regulatory standards, then those opinions would have to be excluded. Ryer-Powder, however, does point to other evidence to support her causation opinion and that is what I turn to next.

Textron argues that Dr. Ryer-Powder's dosage calculation is flawed and artificially high because it failed to take into account a number of contested factual issues: the Woods' use of a water filtration system that would have cut down on the amount of vinyl chloride in the water used for the children and Adele Wood's alleged statements to others that she used bottled water for the children [DE 142 at 22-23; DE 217 at 3-4]. The problem with this argument is that these factual statements are in dispute, and would need to be resolved by a jury. Though Dr. Ryer-Powder would certainly be susceptible to cross-examination on these points, this is not a basis upon which to exclude her testimony entirely.

So this leaves me with the crux of Textron's argument pertaining to Dr. Ryer-Powder: that the scientific studies she has relied upon to come to her causation opinions do not support her conclusions. My task in this case is to "carefully analyze the studies on which experts rely for their opinions before admitting their testimony." *Knight*, 482 F.3d at 355.

As an initial matter, Textron contends that Dr. Ryer-Powder relied principally on "secondary literature," which are articles or reports that recite results found in other studies or secondary literature, as opposed to primary research, or actual reports of scientific studies conducted by the authors [DE 142 at 15]. Textron is correct that courts have treated secondary sources as "simply not scientific evidence" that "purports to rely on scientific studies." *LeBlanc v. Chevron USA, Inc.,* 396 Fed. Appx. 94, 100 (5th Cir. 2010); *see also Aurand*, 802 F. Supp. 2d at 958. In fact, the *LeBlanc* court explicitly rejected one of the secondary sources Dr. Ryer-Powder relied upon in this case, a report of the Agency for Toxic Substances and Disease Registry. *LeBlanc*, 396 Fed. Appx. at 100. Accordingly, these sources alone are not sufficient for an expert to base her testimony upon. However, Dr. Ryer-Powder does cite some primary studies, and I will begin my analysis by looking at those.

In her reports, Dr. Ryer-Powder cites to a study by Maltoni, et al., 1981,[2] to support her contention that an increased cancer risk has been observed in a primary scientific study involving vinyl chloride at dosages relatively close to those present in this case [DE 168-1 at 6-7]. In the study, rats were given vinyl chloride at varying doses: .03, .3, and 1.0 milligrams of vinyl chloride per kilogram of body weight per day. In her report, Dr. Ryer-Powder claims that animals in the control group had 16 tumors per 100 animals; animals in the .03 group experienced tumors in 18 of 100 animals; at the .3 level, there were 13 tumors per 100 animals, and at the 1.0 mg/kg-day dose, there were 24 tumors per 100 animals [*Id.*]. From this data, Dr. Ryer-Powder concludes that there is an "increase in tumors at .03 mg/kg-day," a level of

[2]Cesare Maltoni ET AL., *Carcinogenity Bioassays of Vinyl Chloride Monomer: A Model of Risk Assessment on an Experimental Basis*, 41 ENVTL. HEALTH PERSP. 3 (1981) [DE 142-6].

exposure only ten times greater than that of E.W and C.W., whom Dr. Ryer-Powder opines had exposure levels of .003 mg/kg-day and .002 mg/kg-day, respectively. Dr. Ryer-Powder relied on the Maltoni study as a rejoinder to Textron's arguments that she only relied on high dose studies. She claims that the Maltoni study shows that "exposures to vinyl chloride at a level only ten times greater caused more cancers," and that the study is "directly comparable to the exposures at issue" [DE 168 at 6].

Dr. Ryer-Powder has failed to present the full story of the Maltoni study. The study itself indicates that the lowest dosage of VC administered by ingestion that produced VC-related tumors was .3 mg/kg-day – 100 times greater than the Woods' dose, which was .002 mg/kg-day for C.W. and .003 mg/kg-day for E.W [DE 142-6 at 19, 27]. The study therefore concluded that "[n]one (or no increase) of the specifically VC related tumors . . . was found at doses of 5 and 1 ppm (by inhalation) and .03 mg/kg (by ingestion)" [DE 142-6 at 20]. In other words, the study explicitly found that there was *not* a correlation between increased tumors and the VC levels that were only ten times higher than the doses Dr. Ryer-Powder claims were present in the Woods' case.

What's more, the study explicitly discussed that the data it provided from animals could, at some point, be used in conjunction with human data, to provide "an opportunity, unique at present, to compare animal and human data, both in qualitative and quantitative terms, and to help find a possible key for extrapolating from animals to humans" [DE 142-6 at 25]. In other words, the study expressly did *not* claim that it – in and of itself – provided a basis for extrapolating animal data to humans, but instead indicated that such extrapolation could perhaps

be performed in future studies.[3] That this is the study that the Woods point to in their attempt to bolster Dr. Ryer-Powder's opinion leads me to believe that the basis for her conclusions regarding the effects of vinyl chloride at the levels present in this case is on very flimsy scientific ground.

Another primary study relied upon by Dr. Ryer-Powder was by Drew et al.[4] But the only mention in her report of the Drew study is this odd phrase: "Drew et al (1983) concluded from their studies that exposed were most effective [sic] when started early in life" [DE 147-1 at 17]. Presuming that Dr. Ryer-Powder's point is that exposure to carcinogens is more harmful the earlier the exposure, she fails to explain just how the Drew study supports her conclusions here. A review of the Drew study shows that the purpose of the study was to look at the incidence of tumors that resulted from exposures to chemicals over time in an animal's life, and to determine whether shorter periods of exposures to chemicals would be as effective in producing tumors, so that scientists could conduct more studies of more chemicals in a shorter period of time [DE 142-7 at 2-3]. The study used vinyl chloride to produce tumors because of the availability of data and because "its carcinogenic properties have been extensively studied" [DE 142-7 at 3]. Rats were dosed with vinyl chloride by inhalation at a level of 100 parts per million; mice at 50 parts per

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[3]Dr. Ryer-Powder's rebuttal report also cites a study by Maltoni et al (1984), which she writes "showed that exposure of newborn rats to vinyl chloride by inhalation induced high incidences of liver angiosarcoma and hepatocellular carcinoma. When 11-week old rats were exposed to vinyl chloride under the same conditions, there was almost no carcinognic effect." [DE 168-1 at 8]. The source was not provided to the Court, and Dr. Ryer-Powder has provided no information regarding the dose given to the rats.

[4]Robert T. Drew ET AL., *The Effect of Age and Exposure Duration on Cancer Induction by a Known Carcinogen in Rats, Mice, and Hamsters*, 68 TOXICOLOGY & APPLIED PHARAMACOLOGY 120 (1983) [DE 142-7].

million, and hamsters at 200 parts per million [DE 142-7 at 4]. These doses are thousands of times higher than the (at-most) 9 parts per billion that the Wood children were exposed to – but Ryer-Powder's report makes no mention of this at all, much less attempts to explain how these numbers are applicable to this case.

Ignoring the dose, as Ryer-Powder does, is a critical error. This is because, as one commentator has put it, "the dose makes the poison." Bernard Goldstein and Mary Sue Henifin, *Reference Guide on Toxicology* in *Federal Judicial Reference Manual on Scientific Evidence* at 636 (3d ed. 2011). What this means is that an expert can't simply cite to a study which purports to show that a certain chemical causes harm without evaluating the extent of the exposure. *Id*. All chemicals are potentially hazardous to humans, but whether harm actually results depends upon the level of the exposure. *Id.*

Dr. Ryer-Powder also cites to a primary study by Feron et al., 1981, in which rats were fed vinyl chloride at doses of 0, 1.7, 5.0, or 14.1 mg/kg-day, and all groups showed an increase in mortality and carcinogenic liver lesions [DE 168-1 at 7]. The lowest dose in the Feron study – 1.7 mg/kg-day – is the equivalent of 20 parts per million.[5] And 20 parts per *million* is thousands of times more than the 9 parts per *billion* at which the Wood children were exposed. For Dr. Ryer-Powder's part, she attempts to explain this discrepancy by saying that "[g]iven the sizeable increase in cancerous liver effects from 0 to 1.7 mg/kg-day and the dose-dependent nature of the effects, one can conclude that cancerous effects occur at doses between 1 and 1.7 mg/kg-day"

[5]Though not explained by Dr. Ryer-Powder, Textron indicated at the oral argument on this motion that the human equivalent dose to 1.7 mg/kg-day, per the United States EPA, would be 1.07 mg/kg-day, assuming continuous human exposure over a lifetime, or seventy years. Though this is lower, it still exceeds the dosage present in this case and represents a much longer period of time than the exposure present here.

[DE 168-1 at 8]. However, this conclusion lacks scientific backing, fails to explain how this case applies to the Woods, and represents nothing more than Dr. Ryer-Powder's *ipse dixit. Gen. Electric v. Joiner*, 522 U.S. 136, 146 (1997).

In addition to the Maltoni and Drew studies, Dr. Ryer-Powder also cites a number of secondary sources. For example, she cites an article by Kielhorn, et al.,[6] to support her opinion that VC exposure could cause E.W. and C.W.'s gastrointestinal problems [DE 147-1 at 16]. But the Kielhorn article, which surveyed a number of studies, included information regarding industrial workers exposed to VC at levels "up to" 1,000 parts per million and possibly more, whereas C.W. and E.W. were exposed to VC at a rate of, at most, 9 parts per billion – or approximately 100,000 times lower than the concentrations discussed in the article [DE 142-3 at 2].

The next article that Dr. Ryer-Powder relied upon is by Easter, et al.,[7] which she cites for the proposition that VC is an irritant to the eyes, skin, and mucous membranes, and can therefore irritate the GI system [DE 147-1 at 16]. The article identifies itself as a "brief review of an often extensive literature base," and expressly indicates that it may cite only some references [DE 142-4 at 2]. Though a number of sources are cited at the conclusion of the article, the Easter publication does not report the findings of a scientific study conducted by its authors. Without more analysis by Dr. Ryer-Powder in the form of reference to specific studies or dosages, it is not

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[6]Janet Kielhorn ET AL., *Vinyl Chloride: Still a Cause for Concern*, 108 ENVTL. HEALTH PERSP. 579 (2000) [DE 142–3 at 2-11].

[7]M. D. Easter & R. Von Burg, *Toxicology Update*, 14 J. APPLIED TOXICOLOGY 301 (1994) [DE 142-4].

clear from the Easter article how its information can be reasonably applied to the facts of this case.

Similarly, Dr. Ryer-Powder relies on an article by Swenberg, et al.,[8] to support her contention that VC exposure has caused an unacceptable risk of cancer in the future [DE 147-1 at 17]. While the article does explore the effects of VC exposure in newborn rats versus adult rats, the lowest concentration of VC that the rats were exposed to was 600 parts per million [DE 142-5 at 4], many tens of thousands of times higher than the 9 parts per billion to which E.W. and C.W. were exposed, according to Dr. Ryer-Powder [DE 142 at 17]. This article also summarizes studies conducted by others, and does not report original research by its authors.

The problem with Dr. Ryer-Powder's reliance on these articles is that while it is absolutely true that vinyl chloride can cause very serious health problems, there does not seem to be scientific evidence – at least as presented by Dr. Ryer-Powder – that vinyl chloride at the doses present here can cause the Woods' problems, as she claims. The studies that Dr. Ryer-Powder cites all involve high doses of vinyl chloride, at levels many thousands of times higher than the Wood children experienced. While Dr. Ryer-Powder did not need to cite a study with the exact doses present here, *Daubert* demands that there be some evidence that exposure at levels *somewhat* near those present in this case can cause the Woods' ailments. Alternatively, Dr. Ryer-Powder could have attempted to articulate a scientific basis for applying studies involving high doses, animals, or adults to the situation here. But she failed to do so.

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[8]James A. Swenberg, Norbert Fedtke & Lawrence Fishbein, *Age-related Differences in DNA Adduct Formation and Carcinogenesis of Vinyl Chloride in Rats*, INT'L LIFE SCI. PRESS 163 (1992) [DE 142-5].

To counter Textron's arguments that the bases for Dr. Ryer-Powder's conclusions are too far afield from the facts in this case, the Woods point to three things which they claim enable Dr. Ryer-Powder to extrapolate from the high-dose, occupational, and animal studies: first, they argue that application of occupational and animal studies is permissible, according to Textron's own experts [DE 168 at 7-8, 10]. Second, they contend that E.W. and C.W. have an increased vulnerability to vinyl chloride because they are children [DE 168 at 8]. Finally, the Woods posit that oral consumption results in more vinyl chloride exposure than inhalation [DE 168 at 8-9].

The Woods are correct that "[t]rained experts commonly extrapolate from existing data." *Joiner*, 522 U.S. at 146. Experts look at existing data that may not reflect the same numbers present in the case they are analyzing, but with their training and experience, they are able to apply the published data to different situations. However, under *Daubert*, experts have to connect the dots for the court and explain *how* they extrapolated the data, and if they fail to, "[a] court may conclude that there is simply too great an analytical gap between the data and the opinion proffered." *Joiner*, 522 U.S. at 146. In other words, courts should not admit "opinion evidence that is connected to existing data only by the *ipse dixit* of the expert." *Id.*

In *Joiner*, the Supreme Court upheld a District Court's decision to exclude expert testimony on causation regarding polychlorinated biphenyls (PCBs) and lung cancer. 522 U.S. at 144-145. In that case, the Plaintiff's expert purported to rely on a number of studies indicating a link between PCB exposure and cancer. *Id.* The Defendants objected to the proposed expert testimony, claiming that the studies that the Plaintiff's expert relied upon did not support his opinions, which were simply speculative. *Id.* at 143. The studies at issue in *Joiner* involved infant mice who were directly injected with high concentrations of PCBs and subsequently

developed cancers unlike those that the Plaintiff, an adult exposed to lower concentrations of PCBs by liquid contact with his skin, had developed. *Id.* at 143-144. The Plaintiffs failed to explain "how and why the experts could have extrapolated their opinions from these seemingly far-removed animal studies." *Id.* at 144. Additionally, four other epidemiological studies (meaning studies involving human populations) were not proper bases for the expert's opinion: one involved workers exposed to PCBs but failed to find grounds for "associating lung cancer deaths . . . and exposures at the plant," despite higher rates than would normally be expected; one found that the increase of lung cancer deaths among plant workers exposed to PCBs was elevated, but not statistically significant and did not suggest a link between the increase in deaths and PCB exposures; the third did not pertain to PCBs; and the fourth involved exposure to a number of carcinogens, not just PCBs. *Id.* at 145-46. Accordingly, the Supreme Court found, the analytical gap between the cited studies and the experts' opinions was simply too great to be admissible under *Daubert*.

The Woods' experts are flawed in the same way as the expert was in *Joiner*: the studies upon which they rely do not sufficiently establish that the dose and duration of exposure can cause the injuries complained of in this case, and their bases for extrapolating from studies with dissimilar data are not reliable.

Joiner is essentially dispositive here. As Textron argues, the studies that Dr. Ryer-Powder relied upon in reaching her conclusions as to general causation – in other words, that the type of exposure the Woods experienced can cause the problems they assert – concern animals, worker studies, and much higher dosages. Indeed, as demonstrated above, in some cases the dosage was thousands of times higher than those present in this case. Though the Woods

contend that Dr. Ryer-Powder has extrapolated her results based on scientific evidence, the bottom line is that she has failed to explain her reasoning for extrapolating *these* results, in *this* case.

For instance, as to extrapolating adult exposures to children, she says only that "[s]tudies demonstrate an increased susceptibility to cancer when exposed as a newborn" [DE 168-1 at 5]. Though she cites some secondary sources in support of this contention, she fails to explain *how* studies involving doses many thousands of times greater than the doses in this case can be extrapolated to support her conclusions here. Instead, she simply concludes that "[t]his satisfies general causation, i.e., there is an increased risk to [C.W. and E.W.] to develop cancer in this case as a result of their vinyl chloride exposure" [DE 168-1 at 5]. What's more, she claims that "[t]here is scientific evidence to show that there is a higher sensitivity to liver tumor induction if exposed early in life. Studies on the mechanism of vinyl chloride-induced carcinogenesis support this" [DE 168-1 at 6]. But the question is, what studies, and how do they support application of high dosage studies in this case? Although Ryer-Powder names the studies above, she does no analysis concerning *how* those studies can support the conclusions that she draws in this case – instead, she simply opines that even though the studies that she cites are "not at the same concentration of vinyl chloride . . . the increased vulnerability of children (especially infants and newborns) to toxic injury provides the scientific support to extrapolate to the current case" [DE 168-1 at 6].

The problem here is that while Ryer-Powder cites studies that involve harm from vinyl chloride, and studies that demonstrate that young animals are more susceptible than older animals to harm from vinyl chloride, she doesn't explain *why* or *how* those studies apply here to

bridge the gap – instead, she simply says that it is so, which is the textbook example of an expert relying on her *ipse dixit*. *See Joiner*, 522 U.S. at 146 (". . . nothing in either *Daubert* or the Federal Rules of Evidence requires a district court to admit opinion evidence that is connected to existing data only by the *ipse dixit* of the expert"). There has been no attempt to quantify or give context to just how much more susceptible children are to vinyl chloride. *See, e.g., Baker*, 680 F. Supp. 2d at 876 (where Plaintiffs were exposed to toxins as children, expert calculated an adjusted exposure level based on the EPA's "Supplemental Guidance For Assessing Susceptibility From Early Life Exposures to Carcinogens (2005)"). And given that the dosages in Dr. Ryer-Powder's cited studies are so much greater than the exposures here – in some cases, many thousands of times more – this explanation becomes even more important than if the numbers were close in the first instance.

The same problem befalls the Woods' claim that Dr. Ryer-Powder's extrapolation from high-dose studies is warranted because ingestion is the most potent form of exposure, and most high-dose studies involving occupational settings concern inhalation. Dr. Ryer-Powder's reports provide no basis for this extrapolation (in fact, the Plaintiffs' only support for this argument comes from the deposition of Textron's expert). It may well be true that ingestion of vinyl chloride-contaminated drinking water leads to higher absorption or exposure, but the question then becomes just how much more –many thousands of times more, as would be necessary to bridge the gap between the high dose studies that Ryer-Powder cites and the low level of exposure in this case? As her opinions are presently stated, this question remains unanswered, and under *Joiner*, it means that she has failed to bridge the gap between the existing data and her proffered opinion.

The Woods claim that "Dr. Ryer-Powder extrapolates from animal studies," in the same way that Textron's own experts have [DE 168 at 10]. No one claims that such studies are not relevant, but this precise approach was rejected in *Joiner*:

> Rather than explaining how and why the experts could have extrapolated their opinions from these seemingly far-removed animal studies, respondent chose to proceed as if the only issue was whether animal studies can ever be a proper foundation for an expert's opinion. Of course, whether animal studies can ever be a proper foundation for an expert's opinion was not the issue. The issue was whether *these* experts' opinions were sufficiently supported by the animal studies on which they purported to rely. The studies were so dissimilar to the facts presented in this litigation that it was not an abuse of discretion for the District Court to have rejected the experts' reliance on them.

522 U.S. at 144-45. As in *Joiner*, the Woods have argued only that extrapolation from animal studies can be an appropriate basis upon which an expert may rest her opinion. The flaw, however, is that the Woods have failed to address whether *this* expert's opinion was sufficiently supported by the animal studies on which she relies. There has been no explanation as to how or why a qualified expert could interpret the information regarding animal studies and apply it to humans – a question left open by the very studies upon which Dr. Ryer-Powder relied [DE 142-6 at 25]. At bottom, Dr. Ryer-Powder's report provides no explanation as to how or why these animal studies are applicable to human children.

In sum, as in *Joiner*, the analytical gap between the studies that Dr. Ryer-Powder cites and the facts of this case is just too great. Her opinions must therefore be excluded.

II. Dr. Byers

Dr. Byers has authored an initial report [DE 170-1] and a rebuttal affidavit in response to Textron's motion to exclude her report and testimony [DE 170-4]. In her report (which has no opinion section and doesn't lay out her opinions in a summary manner), Dr. Byers opines that as

a result of the VC exposure, C.W. and E.W. are at an increased risk of cancer, have suffered immune system damage, and have experienced gastrointestinal problems [DE 170 at 1; DE 170-4]. She offers opinions relating to both general and specific causation – specifically, she opines that not only is vinyl chloride *capable* of causing the problems suffered by the Wood children, but that it did in fact cause the problems that she identifies in her report. In her rebuttal report, she claims that Textron's objections to her cancer risk opinion is supported by a number of studies that demonstrate "statistically significant increases in human cancers" [DE 170-4 at 4-5].

Similar to its objections to Dr. Ryer-Powder, Textron argues that Dr. Byers' opinions regarding general causation – that is, that vinyl chloride *can* cause the injuries of which the Woods complain – is not supported by scientific literature, and that the studies that she cites are inapplicable because they concern high-dose, worker exposure studies [DE 155 at 8]. I will address these contentions as they relate to each of Dr. Byers' opinions below.

First, Dr. Byers writes that "[v]inyl chloride has been associated with a wide variety of cancer types" and cites to Vogelstein et al. 2000[9] and Vousden and Land 2007.[10] The Vogelstein article discusses tumor-suppression genes and how their failure to function properly leads to cancer. It makes no mention of vinyl chloride or chlorinated solvents. Similarly, the Vousden article also discusses a protein known as "p53" that plays a role in keeping cancer from occurring. It is also completely devoid of any reference to vinyl chloride or chlorinated solvents.

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[9]Bert Vogelstein, David Lane & Arnold J. Levine, *Surfing the p53 Network*, 408 NATURE 307 (2000) [DE 155-1].

[10]Karen H. Vousden & David P. Lane, *P53 in Health and Disease*, 8 NATURE REVS. 275 (2007) [DE 155-2].

It is difficult to see how these studies support Byers' statement that vinyl chloride has been associated with many types of cancer, given that neither study even mentions vinyl chloride.

Byers also cites a number of worker studies to support her opinions regarding the link between cancer and vinyl chloride. First, she indicates that the signature lesion produced by vinyl chloride is angiosarcoma, citing Creech & Johnson 1974.[11] That article is one-and-a-half-page case report involving one person who worked at a chemical company manufacturing polyvinyl chloride ("PVC") resins.[12] The article mentions at the outset that the authors, who worked for the chemical company, had learned of approximately four patients who had worked at the company's PVC plant and had developed angiosarcomas. No information was given regarding the amount of PVC – or, for that matter, vinyl chloride – to which the workers were exposed, but the main subject had worked at the plant for nearly twenty-five years, and Byers never explains exactly how PVC exposure and dosage relates to VC exposure and dosage.

Gennaro 2008[13] similarly involved industrial workers exposed to PVC compounds, including vinyl chloride. The article concludes that workers exposed to PVC and VC had a statistically significant increase in liver cancer, brain cancer, lung cancer, and tumors of the

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[11]J. L. Creech, Jr. & M. N. Johnson, *Angiosarcoma of Liver in the Manufacture of Polyvinyl Chloride*, 16 J. OCCUPATIONAL MED. 150 (1974) [DE 155-3].

[12]Vinyl chloride monomer is used in a chemical process to produce PVC resins, or plastics [DE 142-3 at 2].

[13]Valero Gennaro ET AL., *Reanalysis of Updated Mortality Among Vinyl and Polyvinyl Chloride Workers: Confirmation of Historical Evidence and New Findings*, BMC PUBLIC HEALTH, 8:21 [DE155-4].

hemolymphopoetic system, but makes no mention of dose [DE 155-4 at 6]. Luo 2003[14] also involved industrial workers, and the study looked at exposures of workers, using a benchmark of 480 parts per million-month to divide workers into high and low exposure groups, and the study specifically cautioned that it may have underestimated the workers' total VC exposure [DE 155-5 at 3-4]. The study also examined workers who were exposed to VC over the course of many years [*Id.*]. Smith 1998[15] looked at French workers exposed to vinyl chloride at levels of less than 500, 501-2500, 2501-5000, and greater than 5000 parts per million. The average exposure level was 3,735 parts per million. Similar to the Luo article, the Smith study cautioned that "no direct vinyl chloride data were available."

Once again, the level of VC exposure by the workers discussed in the Smith article is many thousands of times greater than the exposure in this case. It is impossible to draw any conclusions from those studies regarding causation where the orders of magnitude are so high. The problem, as Textron points out, is that Dr. Byers provides no scientific data that directly supports her conclusion that E.W. and C.W. are at an increased risk for cancer given the much lower rates at which they were exposed to vinyl chloride. Again, there is no question that vinyl chloride is a known human carcinogen that can cause cancer, but the question she has left open is whether vinyl chloride can cause cancer at this dose and this duration.

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[14]Jiin-Chyuan John Luo ET AL., *Molecular Epidemiology of Plasma Oncoproteins in Vinyl Chloride Monomer Workers in Taiwan*, 27 CANCER DETECTION & PREVENTION 94 (2003). [DE 155-5].

[15]Steven J. Smith ET AL., *Molecular Epidemiology of p53 Protein Mutations in Workers Exposed to Vinyl Chloride*, 147 AM. J. EPIDEMIOLOGY 302 (1998) [DE 155-6].

As with Dr. Ryer-Powder, the Woods argue that Dr. Byers is qualified to extrapolate high-dose data to apply to her conclusions here. But just as with Dr. Ryer-Powder, Byers' report is devoid of any explanation of just how she can reliably make this comparison. At her deposition, Dr. Byers was asked to explain how she was able to extrapolate from worker studies and use that information to come to her conclusions regarding the Wood children [DE 128-2 at 28]. She answered flatly that she was able to do so "[b]ecause it's a known human carcinogen and because they were exposed at a very young age" [*Id.*]. She also claimed that it is "accepted that if you are exposed from age zero to age two, your risk is ten times, at least, what it would be if you were an adult" [*Id.*]. She also included this information in her rebuttal affidavit, claiming that ". . . in 2003, the EPA proposed that in determining any risk, regulators should assume children face ten times higher [any risk] than that faced by adults" [DE 170-4 at ¶ 6]. However, this EPA "proposal" was not provided with her report or rebuttal affidavit, nor did Byers indicate if the proposal was ever adopted. Even accepting this ten-fold assumption as true, it is not apparent, given the high dose studies she relies upon, that even a ten-fold increase in the dose the Wood children experienced would put them on par with the studies that Dr. Byers cites. Dr. Byers has not provided any sort of calculations to connect the dots as to how high dose worker studies – some involving doses thousands of times higher than the exposures here – can be extrapolated to support the reliability of Dr. Byers' statements. Without this type of scientific backing, Dr. Byers' opinions regarding dose and causation are simply not reliable, and her opinions regarding the risk of cancer must be excluded.

Dr. Byers also cites a number of worker studies to support her conclusions that E.W and C.W. have immune system damage: Lilis et al. 1975,[16] Ward et al. 1976a,[17] and Ward et al. 1976b.[18] These articles were not provided to the court, but their titles indicate that they involve vinyl chloride and polyvinyl chloride workers. Textron has argued that each of these studies involves industrial workers [DE 155 at 13], and the Woods have not objected to this characterization. The Woods rely upon the same arguments that they raised as to Dr. Ryer-Powder, that Dr. Byers is qualified to extrapolate from these high dose studies because of her training and expertise. There is no evidence of what the dose or duration of exposure was in these studies, so there is simply no way to evaluate whether these studies are even applicable to this case. Accordingly, these opinions are not reliable and cannot establish causation.

Dr. Byers' report does not include a specific section regarding GI problems, but in her discussion of the Wood children's acute symptoms, she has indicated that "[t]he GI problems suffered by both children but most seriously by [E.W.] are consistent with the adverse events associated with vinyl chloride exposure (Compendium of Chemical Hazards/Vinyl Chloride)" [DE 170-1 at 9]. At her deposition, however, she said that she was relying on Dr. Ryer-Powder's opinions regarding the Woods' GI problems [DE 128-2 at 12].

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[16]Ruth Lilis ET AL., *Prevalence of Disease Among Vinyl Chloride and Polyvinyl Chloride Workers*, 246 ANNALS N.Y. ACAD. SCI. 22 (1975).

[17]A. Milford Ward, *Evidence of an Immune Complex Disorder in Vinyl Chloride Workers*, 69 PROC. ROYAL SOC'Y MED. 289 (1976).

[18]A. Milford Ward ET AL., *Immunological Mechanisms in the Pathogenesis of Vinyl Chloride Disease*, 1 BRIT. MED. J. 936 (1976).

As discussed above, the scientific underpinning for Dr. Ryer-Powder's opinion on this issue is weak: the Kielhorn study she cited to support her conclusion that vinyl chloride could cause the Woods' gastrointestinal problems involved industrial workers who were exposed to levels many thousands of times higher than the Woods. Accordingly, this opinion, along with the others of Dr. Byers, must be excluded.

III. Dr. Jeffrey Dahlgren

Dr. Dahlgren submitted an initial report in this matter [DE 146-1], and in response to Textron's motion to exclude his report and testimony, he submitted a "rebuttal declaration" [DE 169-1]. Dr. Dahlgren opines that drinking vinyl chloride-contaminated water has caused the Wood children's gastrointestinal, respiratory, neurological, and immunological problems, and that it is highly likely that they will be diagnosed with cancer at some point in the future [DE 146-1 at 1, 5, 31-34]. Dr. Dahlgren offers opinions relating to both general and specific causation – like Dr. Byers, he opines that not only is vinyl chloride capable of causing the problems suffered by the Wood children, but that it did in fact cause the problems that he identifies in his report. In his rebuttal declaration, he claims to "summarize my prior opinions adding detail and responding to Textron's arguments." [DE 169-1 at 1]. Specifically, he argues that his reliance on studies and research involving trichloroethylene ("TCE") is appropriate because vinyl chloride and TCE are toxicologically similar and VC is actually more dangerous than TCE [*Id.*].

Textron raises similar objections to Dr. Dahlgren's testimony as it raised in regards to Dr. Ryer-Powder and Dr. Byers: among other objections, Textron argues Dr. Dahlgren's methodology is flawed because it relies on exceedance of regulatory screening levels; there is not

scientific literature that supports his opinion that the dose and duration of the Woods' exposure could cause their injuries, and he offers no basis for extrapolating from higher-dose studies [DE 157 at 3, 6]. Additionally, Textron argues that to the extent that Dahlgren has relied upon other experts' opinions that have been excluded, Dahlgren's opinions must also be excluded [DE 157 at 3].

First, as to Dahlgren's reliance on regulatory exceedances, I reach the same conclusions as above, in reference to Dr. Ryer-Powder: to the extent that Dahlgren's opinions are based on exceedance of regulatory standards, they are not admissible. *Cunningham,* 569 F.3d at 674-75 ; *Baker*, 680 F. Supp. 2d at 880; *Allen,* 102 F.3d at 198. However, because his opinions are based on other grounds, this is not dispositive and I will examine the other bases as well.

Dr. Dahlgren's report is replete with references to CVOCs, or Chlorinated Volatile Organic Compounds [DE 146-1 at 1]. Vinyl chloride is one of many CVOCs, and Dr. Dahlgren spends much of his report citing studies regarding trichloroethylene ("TCE"), another CVOC, which he claims is "toxicologically relevant because it has a similar mechanism of toxicity for non-cancer endpoints" [DE 146-1 at 3]. Dr. Dahlgren claims that vinyl chloride occurs as a breakdown product in groundwater contaminated with TCE, but that VC is "much more toxic" [*Id.*]. Because of this, he claims, studies involving TCE are applicable here, though there is no evidence that the Wood children were exposed to TCE [*Id.*]. In fact, Dahlgren writes in his report that "TCE was probably present in significant quantities in the decades prior [to the Wood children's exposure], it was probably mostly converted to VC by microorganisms within the soil by the time [C.W. and E.W.] moved in the residence" [DE 146-1 at 6-7]. Regardless of his concession that the Wood children never even lived in the home while TCE was present (if it

ever was, and there is no evidence of that whatsoever), Dahlgren argues over and over that TCE is similar enough to VC that studies regarding TCE – the most well-known of which are from Woburn, Massachusetts, the site of the toxic tort case that was the subject of the book and film *A Civil Action* – are applicable here.

Textron's memorandum in support of its motion to exclude Dahlgren's testimony fails to attack any of the approximately seventy studies that Dahlgren relied upon in his initial report; instead, it argues that Dahlgren admitted at his deposition that "not a single study shows infant children sustaining any injury due to vinyl chloride exposure at the dose and for the duration these Plaintiffs experienced. This shortcoming is fatal to the admissibility of Dahlgren's expert testimony and opinions . . . " [DE 157 at 12]. This argument is not strictly true – it's wholly unsurprising that Dahlgren was unable to cite a study at the precise dose and duration that the Woods children were subject to, and nothing in the case law says that he must do so (and Textron admits as much in their other briefing on these experts). As the Woods argue in their response, and as we are all aware by now, experts can and frequently apply the results of similar studies to new cases and extrapolate the results. *See Joiner*, 522 U.S. at 146. The problem again, however, is that the studies that Dahlgren does cite leave an "analytical gap" between their data and the facts of this case, and Dahlgren fails to bridge that gap by explaining how he is able to extrapolate those results.

The Woods responded to this attack on Dahlgren by citing five particular studies that he relied upon that they claim directly support his opinions and are "directly in comparison with the concentrations at issue in this case" because they involve "exposure to CVOCs in the parts per billion range," starting as low as 1 part per billion [DE 169 at 8]. And again, the Woods have

countered that Dr. Dahlgren is qualified and able to extrapolate the results of higher-dose studies because children are more susceptible to the negative health effects of vinyl chloride exposure and because more vinyl chloride is absorbed into the body via the oral consumption route than by inhalation, which is generally at issue in worker studies [DE 169 at 9]. In their response, they attempt to shore up Dahlgren's reliance on studies relating to TCE, as opposed to vinyl chloride, arguing that they are reliable "because of the toxicological similarities between the toxins" [DE 169 at 8]. They leave all explanation of what that actually means for Dahlgren himself, who gives a lengthy defense in his "rebuttal" declaration. However, a review of the five studies that Plaintiffs point to as directly supporting Dahlgren's opinions is telling.

Burg 1995[19] is a study of individuals exposed to long-term, low-doses of TCE [DE 198-1]. Putting aside the question of whether TCE and vinyl chloride are so similar that TCE research is applicable here, the study does not support Dr. Dahlgren's conclusions. First, the study involved 4,280 people who lived in areas exposed to TCE. While 290 of the 883 households that were studied had exposure ranging from .4 parts per billion to 9 parts per billion (i.e. similar to the exposure at the Wood home), 593 households had exposure in the 14 parts per billion to 234 parts per billion range. The study did not break out results by exposure levels on an individual level, and did not compare health effects to varying levels of exposure. Moreover, the study explicitly states that its results "do not identify a causal relationship between TCE exposure and adverse health effects" and that "[t]his study does not support a cause and effect relationship between TCE exposure and human health outcomes." In addition to explicitly

[19]Jeanne R. Burg ET AL., *The National Exposure Registry – Morbidity Analysis of Noncancer Outcomes from the Trichloroethylene Subregistry Baseline Data*, 4 INT'L J. OCCUPATIONAL MED. & TOXICOLOGY 237 (1995). [DE 198-1 at 2].

disclaiming a causal relationship, the study explicitly outlined many of the problems that have been identified with all of the Woods' experts' testimony:

> The reported literature has many limitations: case reports of human poisonings and occupational studies usually involve exposure levels much higher than those reported in environmental exposures; high-dose animal studies might not be relevant to humans; and human health studies often lack sufficient exposure characterization, controls for important confounding factors, and sample sizes large enough to investigate low-dose effects. These and other limitations must be considered by the reader.

Despite these warnings, none of these caveats are considered by Dr. Dahlgren in his analysis. This study simply does not support his conclusions.

Second, the Feldman 1988[20] study was designed to "assess possible residual neurotoxic effects of chronic environmental exposure to TCE" [DE 198-1 at 25]. The study involved an examination of blink reflex, an objective indicator of the neurotoxic effects of TCE in members of households in Woburn, Massachusetts who drank water that was contaminated with the chemicals trichloroethylene (11-256 parts per billion), tetrachloroethylene (24-26 parts per billion), and 1.2-trans-dichloroethylene (5 parts per billion). According to the authors of the study, those levels of contaminants were thirty to eighty times higher than the EPA's Maximum Contaminant Levels (MCL) for each of the contaminants. By contrast, the exposure here was of a single chemical and was at or below the MCL levels for vinyl chloride. That means that the exposure levels in this case were less toxic and severe than the levels assessed in the Feldman study. Accordingly, it is difficult to see how the Feldman study applies in this case – even assuming that TCE and VC have similar properties.

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[20]Robert G. Feldman ET AL., *Blink Reflex Latency after Exposure to Trichloroethylene in Well Water*, 43 ARCHIVES ENVTL. HEALTH 143 (1988). [DE 198-1 at 23].

Moreover, the individuals in the Feldman study (that is, the residents of Woburn, Massachusetts, who were a well-studied group and the subject of a number of the studies on which Dr. Dahlgren relies), were exposed to numerous potential carcinogens, not just TCE, *and* they were not exposed to vinyl chloride, the only substance to which the Wood children have been exposed. Courts have found that studies involving populations exposed to multiple toxins that are not the subject of the plaintiffs' injuries to be unavailing. *See Joiner*, 522 U.S. at 146 (studies "of no help" where subjects "had been exposed to numerous potential carcinogens," failed to mention the toxin at issue in the case, and were "expressly limited" to a toxin not at issue); *Amorgianos v. Nat'l. Railroad Pass. Corp.*, 303 F.3d 256, 270 (2d Cir. 2002) (expert's opinion not admissible where expert relied upon articles that "involved individuals who were exposed to a variety of solvents, many of which were not contained in the paint [the plaintiff] used").

The Lagakos 1986[21] study also analyzed the Woburn, Massachusetts population, and reported that the users of polluted wells were exposed to TCE (267 parts per billion), tetrachloroethylene (21 parts per billion) and chloroform (12 parts per billion). Months after the wells were shut down, they also showed contamination with trichlorotrifluoroethane (23 parts per billion) and dichloroethylene (28 parts per billion). The study, reported in the Journal of the American Statistical Association, analyzed available data on the incidences of disease in the areas affected by the contaminated well to determine statistical associations between access to the water and increased childhood illnesses (namely leukemia). For the reasons above pertaining

[21]S. W. Lagakos ET AL., *An Analysis of Contaminated Well Water and Health Effects in Woburn, Massachusetts*, 81 J. AM. STAT. ASS'N 583 (1986). [DE 198-1 at 38].

to the Feldman study, the Lagakos study does not support Dr. Dahlgren's opinions: the individuals studied were exposed to numerous potential carcinogens, not just TCE, and were not exposed to vinyl chloride – which is the only substance at issue in this case. *See Joiner*, 522 U.S. at 146; *Amorgianos,* 303 F.3d at 270. And just as with the Feldman study, Dr. Dahlgren fails to explain these differences.

The Cohn 1994[22] study arguably comes close on the dose front, because it looked at the incidence of non-Hodgkin's lymphoma and leukemia in 75 New Jersey towns with some drinking water contamination involving TCE and PCE contamination at a rate of above 5 parts per billion. The authors of the study wrote that "[t]he results suggest a link between TCE/PCE and leukemia/NHL incidence." However, they cautioned, the conclusions were "limited" by potential misclassification of exposure, lack of information on long-term residence, water consumption, and the inhalation of "volatilized compounds." Moreover, the study cautioned that "[t]he carcinogenic activity of TCE and PCE may be compounded by joint exposure because TCE and PCE appear to share toxic metabolic pathways." In any event, the study did not give information on levels of exposure above 5 parts per billion – in other words, there is no information regarding just how high those exposures were, so it is difficult to assess when these chemicals began causing cancers. And as noted with these other studies, there are multiple contaminants present in this study, and none of them are vinyl chloride.

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[22]Perry Cohn ET AL., *Drinking Water Contamination and the Incidence of Leukemia and Non-Hodgkin's Lymphoma*, 102 ENVTL. HEALTH PERSP. 556 (1994). [DE 198-1 at 54].

The Kilburn 2002[23] study involved groundwater contamination in Arizona and studied residents who had lived near electronics plants for two to thirty-seven years. The groundwater was tested and found to contain TCE, TCA, 1,1-DCE, 1,2-DCE, PCE, and VC. TCE was present at levels of less than .2 parts per billion to over 10,000 parts per billion and VC was present at levels from less than .02 parts per billion to 330 parts per billion. The study noted that TCE and TCA "were distributed widely" and that "the wells contained many chlorinated solvents, among which TCE predominated." The study described itself as looking at "long-lasting effects from nearly continuous low level exposures to TCE for many years," not as analyzing any effects of vinyl chloride.

These studies simply do not provide the support that Dahlgren argues that they do: even accepting Dahlgren's representations that TCE and VC are toxicologically similar and TCE research is even applicable to this case, these studies either involve a mix of TCE and other non-VC toxins, or they expressly disclaim a causal relationship between TCE and human health problems. These articles cannot support Dr. Dahlgren's opinions, and fail to establish that VC at the dose and duration present in this case could cause the problems that the Plaintiffs have experienced or claim that they are likely to experience.

* * *

In excluding these experts, I am mindful that "an expert's opinion does not have to be unequivocally supported by epidemiological studies in order to be admissible under *Daubert*." *Baker*, 680 F. Supp. 2d at 887 (citing *Knight*, 482 F.3d at 354). However, in this case, the

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[23]Kaye H. Kilburn, *Is Neurotoxicity Associated with Environmental Trichloroethylene (TCE)?*, 57 ARCHIVES ENVTL. HEALTH 113 (2002). [DE 198-1 at 29].

Plaintiffs' experts have simply not presented reliable bases that support opinions that vinyl chloride, in the dose and duration to which the Wood children were exposed, can cause the problems that they have experienced. The subjects of the studies that were cited almost all had much, much higher exposures to vinyl chloride than the Wood children faced, or exposures to completely different toxins, and the stated bases for extrapolating from these studies essentially boil down to the *ipse dixit* of the experts and of Plaintiffs' counsel. While it is clear from the literature that vinyl chloride is a dangerous chemical that can cause a host of health problems, including cancer, there is simply not evidence that the dose and duration of exposure present here significantly increases the risk of the Plaintiffs' medical problems. Therefore, I find the causation opinions of Drs. Ryer-Powder, Byers, and Dahlgren to be unreliable under *Daubert*, and consequently, inadmissible. Accordingly, Textron's motions to exclude the testimony and reports of Dr. Ryer-Powder [DE 139], Dr. Byers [DE 154], and Dr. Dahlgren [DE 156] are **GRANTED**.

CONCLUSION

For the reasons above, Textron's motions to exclude the testimony and reports of Dr. Ryer-Powder [DE 139], Dr. Byers [DE 154], and Dr. Dahlgren [DE 156] are **GRANTED**. Textron's motion to strike [DE 212] is **DENIED AS MOOT**. Because of the significant impact of these rulings on the future of this litigation, I hereby **DENY AS MOOT** the other pending motions to exclude experts [DE 124; DE 129; DE 130; DE 132; DE 134]. In light of this ruling, I will also **GRANT** Textron's Motion for Leave to File Exhibits Under Seal [DE 218]; however, I also **DENY AS MOOT** the motion itself [DE 217].

The parties are granted until April 15, 2014 to file a status report indicating how they wish to proceed, whether the Court needs to consider the other *Daubert* motions, and whether they anticipate the filing of summary judgment motions. If for whatever reason the parties believe it is necessary for me to decide the other *Daubert* motions, they can so advise me and I will reinstate them to the docket.

SO ORDERED.

ENTERED: March 17, 2014

s/ Philip P. Simon
PHILIP P. SIMON, CHIEF JUDGE
UNITED STATES DISTRICT COURT